**UNITED STATES DISTRICT COURT**
**FOR THE DISTRICT OF NEW JERSEY**
**CAMDEN VICINAGE**

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| **IN RE: VALSARTAN, LOSARTAN, AND IRBESARTAN PRODUCTS LIABILITY LITIGATION**<br><br>**This Document Relates to All Actions** | MDL No. 2875<br><br>Honorable Robert B. Kugler, District Court Judge<br><br>Oral Argument Requested |

# MEMORANDUM OF LAW IN SUPPORT OF DEFENDANTS' JOINT MOTION TO EXCLUDE THE OPINIONS OF MAHYAR ETMINAN, PHARMD, MSC

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**Rules**

Defendants Executive Committee, on behalf of all Defendants in this litigation, respectfully submit this Memorandum of Law in support of Defendants' Joint Motion to Exclude the Opinions of Mahyar Etminan, PharmD, MSc, pursuant to Federal Rules of Evidence 702, 403, and 104.

## INTRODUCTION

Plaintiffs retained Dr. Mahyar Etminan as their putative epidemiology expert to opine on whether N-Nitrosodimethylamine (NDMA) and N-Nitrosodiethylamine (NDEA) in the recalled valsartan products can increase the risk of the following types of cancer: (1) esophageal, (2) stomach, (3) colorectal, (4) liver, (5) pancreas, (6) lung, (7) bladder, (8) prostate and (9) blood (leukemia, lymphoma and multiple myeloma). *See* Ex. A, Expert Report of Dr. Mahyar Etminan, PharmD, MSc (Epidemiology), dated July 6, 2021 ("Report"), at 3. Dr. Etminan's opinions do not satisfy the clear requirements of Rule 702 and established precedent and should be excluded pursuant to Rule 702 of the Federal Rules of Evidence for four principal reasons:

- ***First***, Dr. Etminan's opinions do not "fit" this case because they do not consider the substance, dose, and duration of the exposure in relation to the particular conditions at issue. Here, Plaintiffs allege they were exposed to NDMA and/or NDEA by ingesting valsartan between 2012 through 2018. Dr. Etminan's opinions broadly sweep across all types of NDMA or NDEA

exposure, at any exposure level, for any period of time—circumstances that do not align in any way with this case;

- ***Second***, Dr. Etminan employed an unreliable methodology, which included: 1) failing to account for baseline NDMA or NDEA exposure from endogenous (internally generated) and exogenous (external) sources; 2) purposefully excluding studies from his literature search that show no statistically significant association between valsartan and cancer; and 3) including studies that should have been excluded by his stated literature search criteria;
- ***Third***, Dr. Etminan's opinions do not show a reliable causal relationship based on the epidemiologic data: 1) his methodology ignores widely accepted standards for statistical significance; 2) he applies his own criteria inconsistently in a results-driven manner by relying upon studies when they support his conclusions and criticizing the same studies when they contradict them; and 3) he misapplies the Bradford Hill criteria epidemiologists use to distinguish a causal connection from a mere association; and
- ***Fourth***, Dr. Etminan's opinions with respect to NDEA are entirely unsupported by scientific evidence, as are his opinions with respect to NDMA's alleged causal role in certain cancers. Accordingly, at a minimum, the Court should exclude these unreliable opinions.

For these reasons, Defendants respectfully request that the Court exclude Dr. Etminan's opinions.

## SUMMARY OF OPINIONS TO BE EXCLUDED

Defendants seek exclusion of the following opinions disclosed by Dr. Etminan:

- Any opinions concerning a supposed causal association between NDMA and Esophageal,[1] Stomach, Colon, Liver, Pancreas, Lung, Bladder, Prostate, And Blood (Leukemia, Lymphoma Multiple Myeloma) Cancers, (Report at 32);

- Any opinions concerning a supposed causal association between NDEA and Esophageal, Stomach, Colon, Liver, Pancreas, Lung, Bladder, Prostate, And Blood (Leukemia, Lymphoma Multiple Myeloma) Cancers, (Report at 31-32); and

- Any opinions concerning statistical significance or the statistical significance of any particular study finding (*see* Report at 12) (disregarding accepted standards on statistical significance and

[1] It is unclear whether Dr. Etminan is offering a causation opinion only on esophageal cancer or on "head and neck" cancers, including pharyngeal and laryngeal, as a group. *Compare* Report at 3 (opining only on esophageal cancer), 32 (concluding NDMA causes esophageal but not laryngeal or pharyngeal cancer), *with* Report at 20-21 (concluding "it is probable that high exposure to NDMA increases the risk of head and neck cancers"). However, Dr. Etminan should be precluded from offering any opinions as to pharyngeal or laryngeal cancers. *See infra* at 24-33. (discussing basis for excluding Dr. Etminan's unsupported causation opinions). Dr. Etminan does not cite a single study providing support for a causal association between pharyngeal cancer and NDMA/NDEA. *Compare* Report at 20-21 (failing to mention any studies examining pharyngeal cancer) *with* Report at 23 (noting *Straif*'s lack of finding on pharyngeal cancer). Nor does he cite any study specifically examining NDMA and only laryngeal cancer that showed a significant association. *See* Report at 20-21.

asserting that a study's results are not "necessarily negative" where its confidence interval is inclusive of 1.0).

## FACTUAL AND PROCEDURAL BACKGROUND

Defendants hereby adopt and expressly incorporate by reference the "Factual and Procedural Background" set forth in the Memorandum of Law in Support of Defendants' Joint Motion to Exclude the Opinions of Stephen Hecht, Ph.D.

## LEGAL STANDARDS

In addition to the legal standards set forth herein, Defendants incorporate by reference the section entitled "Legal Standards" set forth in the Memorandum of Law in Support of Defendants' Joint Motion to Exclude the Opinions of Stephen Hecht, Ph.D.

Under Federal Rule of Evidence 702, this Court performs a "gatekeeping function" to ensure that all expert testimony is both relevant and reliable. *See In re Paulsboro Derailment Cases*, 746 Fed. App'x 94, 98 (3d Cir. 2018) (Vanaskie, J.) (citing *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 589 (1993)). It is Plaintiffs' burden alone to show Dr. Panigrahy's testimony is admissible. *See Warren Distributing Co. v. Inbev USA L.L.C*, 2010 WL 2179167, at *3 (D.N.J. May 26, 2010) (Kugler, J.) (citing *Kannankeril v. Terminix Int'l, Inc.*, 128 F.3d 802, 806 (3d Cir. 1997)).

"Rule 702 embodies a trilogy of restrictions on expert testimony: ***qualification***, ***reliability***, and ***fit***." *Ruggiero v. Yamaha Motor Corp., U.S.A.*, 778 F.

App'x 88, 93 (3d Cir. 2019) (emphasis added) (quoting *Schneider ex rel. Estate of Schneider v. Fried*, 320 F.3d 396, 404 (3d Cir. 2003)). First, the Court must consider whether the expert is qualified "to render an opinion" based on his or her "specialized expertise." *In re Hum. Tissue Prods. Liability Litig.*, 582 F. Supp. 2d 644, 655 (D.N.J. 2008) (quoting *Pineda*, 520 F.3d at 244). Though qualification is interpreted liberally, the Third Circuit recognizes an expert who "may be generally qualified" may nevertheless "lack qualifications to testify outside his area of expertise." *Calhoun v. Yamaha Motor Corp., U.S.A.*, 350 F.3d 316, 322 (3d Cir. 2003). Second, the Court must evaluate the reliability of the expert's methodology. For a methodology to be reliable, the expert's opinions must be based on methods and procedures of science rather than on subjective belief or unsupported speculation. The expert must have "good grounds" for his or her belief. *See In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 732 (3d Cir. 1994) (quoting *Daubert*, 509 U.S. 579 at 590). Third, the Court must consider whether the expert's testimony will be helpful to the trier of fact. *See* Fed. R. Evid. 702(a). "The issue of fit 'is one of relevance and expert evidence which does not relate to an issue in the case is not helpful.' . . . The standard for fitness is 'not that high' but is 'higher than bare relevance.'" *In re Hum. Tissue Prod. Liab. Litig.*, 582 F. Supp. 2d at 657 (quoting *In re TMI Litig.*, 193 F.3d 613, 670 (3d Cir. 1999); *In re Paoli*, 35 F.3d at 745). An opinion fits and helps the trier of fact when there is a connection between the

scientific research or test result presented and the particular disputed factual issues in the case. *Warren Distributing Co.*, 2010 WL 2179167, at *4.

## ARGUMENT

### I. DR. ETMINAN'S UNSUPPORTED OPINIONS WILL NOT ASSIST THE TRIER OF FACT.

As a threshold matter, Dr. Etminan's putative "general causation" opinions will not assist the Court or a jury because they do not fit the parameters of a general causation opinion that would be meaningful to the facts of this case. An expert's opinions are not admissible if they do not "fit" the case, irrespective of the expert's qualifications and methodology. *In re Diet Drugs Prod. Liab. Litig.*, 706 F. 3d 217, n.7 (3d Cir. 2013). "[I]n a toxic tort case, expert testimony on the issue of general causation meets Rule 702's 'fit' requirement only if the testimony includes an opinion that (1) exposure to the particular substance at issue, (2) in the dose to which the plaintiff was exposed, (3) for the duration in which plaintiff was exposed, (4) can cause the particular condition(s) of which the plaintiff complains." *Amorgianos v. Nat'l R.R. Passenger Corp.*, 137 F. Supp. 2d 147, 163 (E.D.N.Y. 2001), *aff'd,* 303 F.3d 256 (2d Cir. 2002). "Thus, even if an expert's proposed testimony constitutes scientific knowledge, his or her testimony will be excluded if it is not scientific knowledge *for purposes of the case*," and will be precluded. *In re Paoli R.R. Yard PCB Litig.*, 35 F. 3d 717, 743 (3d Cir. 1994); *see also* Memorandum of Law in

Support of Defendants' Joint Motion to Exclude the Opinions of Stephen Hecht, Ph.D. ("Motion to Exclude Hecht"), at section I(A)(3).

Here, Plaintiffs allege they were exposed to NDMA and/or NDEA by orally ingesting valsartan containing trace amounts of NDMA or NDEA impurities measured by the FDA to be no more than 20.1 micrograms in the highest dose tablet, at some point between 2012 through 2018. Dr. Etminan's unsupported opinions for each cancer on which he opines do not fit the facts of this case because he did not assess whether the NDMA and NDEA found in valsartan, at the potential exposure levels identified for the potential duration of time at issue, can cause these cancers. Instead, he was asked generally to "infer whether prolonged NDMA exposure" ***through any method of exposure, at any exposure level***, can cause cancer:

> Q: Okay. For these occupational studies, the method of exposure was primarily through inhalation or skin contact. Would you agree with that?
>
> MR. NIGH: Form objection.
>
> THE WITNESS: Yeah.
>
> BY MR. GALLAGHER: Q: Okay. And that method of exposure is different from the method of exposure that's at issue in this case, correct?
>
> A: The method of exposure is different, but my -- the question, the general causation question that I -- that my report refers to does not specify cause of cancer with NDMA with respect to different rounds of exposure. It refers to, generally speaking, exposure. And we mean systemic exposure, which could be mouth or through the skin or through inhalation cause cancer.

> Q: Okay. So the question that you were evaluating was not whether NDMA ingested orally could cause certain cancers, is that correct?
>
> MR. NIGH: Form objection.
>
> THE WITNESS: No, that's not – that's not what I said. The question that I addressed was: Does exposure to NDMA and exposure would mean NDMA that gets in to the body systemic – systemically absorbed NDMA, which can be through oral, inhalation, skin. I think mainly those are the – the main routes of the exposure. Does builds – does exposure to NDMA through any of those routes that make it systemic in the body cause cancer.

Tr. Vol. I, 55:13-56:19; *see also* Report at 9.

Dr. Etminan's proffered opinions do not fit this case because he failed to focus his analysis on the particular exposure alleged by Plaintiffs — *i.e.* oral ingestion of valsartan tablets containing trace amounts of NDMA/NDEA over a specific alleged duration. *See Hardeman v. Monsanto Co.*, 997 F.3d 941, 963 (9th Cir. 2021) (general causation showing depends upon "exposure levels people realistically may have experienced"); *In re Zoloft (Sertralinehydrochloride) Prod. Liab. Litig.*, 176 F. Supp. 3d 483, 487 (E.D. Pa. 2016), *aff'd,* 858 F.3d 787 (3d Cir. 2017) (framing the general causation question as whether the drug at issue "at therapeutic dose levels" could cause outcome); *In re Viagra (Sildenafil Citrate) & Cialis (Tadalafil) Prod. Liab. Litig.*, 424 F. Supp. 3d 781, 793 (N.D. Cal. 2020) (quoting *In re Hanford Nuclear Rsrv. Litig.*, 292 F.3d 1124, 1133 (9th Cir. 2002)) (general causation question dependent upon "the level of exposure alleged by plaintiffs"); *see also* Motion to Exclude Hecht, at section I(A)(3).

Rather than focus his general causation inquiry on the alleged methods of exposure, amounts of exposure, or duration of exposure at issue in this litigation, Dr. Etminan assumes that ***all types of NDMA or NDEA exposure***, ***at any dose level, for any period of time***, translate to this case. As Dr. Etminan's opinions do not answer the appropriate general causation question evaluating what level of ingested NDMA or NDEA Plaintiffs might reasonably have been exposed to under the potential limited time frame, they do not "fit" the facts of this case as required by Rule 702 and must be excluded on this threshold basis.

## II. DR. ETMINAN'S OPINION SHOULD BE EXCLUDED BECAUSE HE DID NOT EMPLOY A RELIABLE METHODOLOGY.

Dr. Etminan did not reach his conclusions through use of reliable methodology or data and, therefore, his opinions should be excluded under Rule 702. *See* Fed. R. Evid. 702(c) (requiring "the testimony [to be] the product of reliable principles and methods"). In evaluating the reliability of an expert opinion, courts are advised "to determine whether the analysis undergirding the experts' testimony falls within the range of accepted standards governing how scientists conduct their research and reach their conclusions." *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995); *see also Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 590 (1993) ("Proposed testimony must be supported by appropriate validation—*i.e.*, 'good grounds,' based on what is known.").

"[S]ound scientific methodology requires that a scientist consider all of the scientific evidence when making causation determinations." *See In re Zoloft (Sertraline Hydrochloride) Prod. Liab. Litig.*, 858 F.3d 787, 799-800 (3d Cir. 2017). Dr. Etminan failed to follow sound scientific methodology and neglected to consider all the scientific evidence, instead selectively including and excluding data to promote his predetermined conclusions. That is not science, and his opinions cannot be admitted under Rule 702 and *Daubert*.

### A. Dr. Etminan Does Not Calculate Or Consider Baseline NDMA Or NDEA Exposure.

By Dr. Etminan's own admission, NDMA and NDEA are ubiquitous substances in the environment to which all humans are exposed at some level. *See* Report at 31 ("[I]t is impossible to identify subjects with absolutely no exposure to NDMA as this agent is ubiquitous in the environment and thus some level of exposure is always expected."); *see also* Ex. C, Tr. Vol. II, 14:6-9 ("Q. Every plaintiff in this litigation has been exposed to NDMA and NDEA throughout their lifetimes just like you and I have, right? A. Yes."). As reflected in a study Dr. Etminan cites in his report, this exposure may come from internal, or endogenous, processes within the body, or from external, or exogenous, sources such as the air, water, or food. *See, e.g.*, Ex. D, Jakszyn *et al.* (2006); Report at n.47.

Dr. Etminan also admits that further studies are required to establish a baseline level of exposure to NDMA and NDEA from which one could calculate whether an

increased risk of cancer exists from the amounts of NDMA and NDEA contained in valsartan. *See* Tr. Vol. II, 19:24-20:8 ("Q. And I trust you'd agree with me that if you want to evaluate the impact of nitrosamines in valsartan-containing medications, what we need to consider is the extent to which individual consumption of NDMA and NDEA increase due to the presence of those compounds in the drugs, right? MR. NIGH: Form objection. THE WITNESS: I mean, if you want to do a perfect study, yes, that's – that's what needs to be done.").

Despite these admissions, Dr. Etminan did not attempt to quantify or calculate baseline exposure before concluding that the amount of NDMA and NDEA in valsartan causes each of the specific cancers at issue. *See* Tr. Vol. II, 19:18-23 ("Q. And as part of your work in this case, you have not done any independent research studies or testing to – to suggest or establish that the estimates of total nitrosamine exposure as predicted by Jakszyn were incorrect, fair to say? A. Yes."). In fact, Dr. Etminan did not consider endogenous formation of NDMA at all in rendering his opinions. *See* Ex. B, Tr. Vol. I, 113:17-114:1 ("Q. Do you have an understanding of the distinction between endogenous NDMA and exogenous NDMA? A. To – to – you know, to certain levels, yes. Q. Okay. You haven't addressed anywhere in your report the – the – any potential impact of endogenous NDMA, have you? MR. NIGH: Form objection. THE WITNESS: No[.]"). He did not consider endogenous formation of NDMA despite citing to articles in his report that, when accounting for

endogenous *N*-nitroso compounds, determined there was no association of exogenous NDMA exposure and gastric cancer. *See* Ex. D, Jakszyn *et al.* (2006); Tr. Vol. I, 122:16-123:17. If, as Dr. Jakszyn's peer-reviewed study reports, the level of endogenous production of NDMA is higher than the levels reported in the valsartan products, Dr. Etminan's opinions that the trace levels in valsartan cause cancer are unfounded and illogical.

Having failed to establish a baseline level of NDMA or NDEA exposure, Dr. Etminan likewise made no attempt to analyze whether an incremental increase in NDMA or NDEA exposure over a limited period of time would lead to a statistically significant increased risk of developing cancer given the human population's significant background nitrosamine exposure. Tr. Vol. II, 30:4-31:14; 41:21-42:1. Further, he could not cite to a single study in the scientific literature that attempted to analyze this issue. *Id.* at 37:3-20.

It is impossible for Dr. Etminan to assess whether the amounts of NDMA or NDEA in valsartan could even increase the risk of any specific cancer over the general population's risk without considering the baseline levels of NDMA or NDEA to which the general population is likely exposed. *See Soldo v. Sandoz Pharms. Corp.*, 244 F. Supp. 2d 434, 533 (W.D. Pa. 2003) (finding that, where a disease occurs in the general population, "[t]he need for statistically significant epidemiology is particularly acute" in order "to determine whether any given case

of [the disease] could possibly be attributable to a particular drug."). Dr. Etminan's total failure to consider baseline exposures is thus a fundamental flaw in his methodology.

### B. Dr. Etminan's Methodology Is Unreliable Because His Literature Search Criteria Excluded The Only Studies Evaluating Valsartan Containing NDMA Impurities—The Specific Exposure At Issue In This Case.

Dr. Etminan's methodology excludes not only baseline exposures, but also highly relevant scientific literature. Although Dr. Etminan claims to have conducted a systematic literature search, in reality, his inclusion and exclusion criteria were outcome-based and inherently biased because they were designed to exclude the most relevant epidemiologic literature: the Gomm and Pottegard studies. *See* Ex. E, Gomm *et al.*; Ex. F, Pottegard *et al*. Those studies specifically tested for a possible association between valsartan containing NDMA impurities and cancer, and found none. If an expert does not sufficiently discredit or meaningfully address alternative explanations for studies that find no association or no statistically significant association between the disease and the exposure, the reliability of the expert's opinion is undermined. *See In re Zoloft (Sertraline Hydrochloride) Prod. Liab. Litig.*, 858 F.3d 787, 799-800 (3d Cir. 2017). Likewise, "[a] district court may properly consider the fact that an expert relied on few, if any, studies linking exposure to a particular illness in evaluating whether the expert had 'good grounds'

to arrive at his conclusions." *Pritchard v. Dow Agro Scis.*, 705 F. Supp. 2d 471, 485 (W.D. Pa. 2010), *aff'd,* 430 F. App'x 102 (3d Cir. 2011).

The Gomm and Pottegard studies examined the risk of cancer in thousands of German and Danish patients, respectively, who had consumed prescribed valsartan that potentially contained NDMA. These studies used comprehensive data from healthcare registries to classify patients based on potential exposure to NDMA, and followed those subjects until death or the end of the study. ***Both studies observed no statistically significant increased risk of cancer due to NDMA-containing valsartan***. *See* Gomm *et al.* and Pottegard *et al.*, Exs. E and F.

While Dr. Etminan's literature search included "[s]tudies that looked at valsartan containing NDMA or NDEA formulations," he placed a qualifier on this inclusion criterion in order to exclude the Gomm and Pottegard studies, the only two available valsartan studies. Specifically, Dr. Etminan required that a study "had to be able to differentiate NDMA or NDEA specific valsartan batches (from batches that did not contain excessive amounts of NDMA or NDEA)" before he would consider it. Report at 13. The Gomm and Pottegard studies both considered the NDMA exposure for cohort participants based on cumulative dose of valsartan but did not track each participants' intake by specific manufacturers' batches. Report at 26. There is no scientific reason for this artificial limitation. By imposing this arbitrary requirement, Dr. Etminan excluded relevant studies demonstrating no

statistically significant increased risk of cancer from NDMA in valsartan, in favor of less relevant diet and occupational studies, many of which also demonstrated a lack of statistical significance for an association between NDMA and the cancers Dr. Etminan claims to be addressing. Because Dr. Etminan's literature search criteria were designed to exclude the most relevant data available to the general causation inquiry, his methodology is unreliable.

### C. Dr. Etminan Improperly Relies On The Hidajat Occupational Study, Which Should Have Been Excluded By His Own Search Criteria.

Although Dr. Etminan claims to have reviewed the "totality of the evidence," he largely relies on one occupational study, the Hidajat study, to support his opinions on eight of the nine cancers addressed in his Report. Tr. Vol. I, 193:21-194:9 ("Q: Okay. When you say 'they are similar in design,' I thought we had discussed earlier this morning, that all of the studies have different designs, right? A: Well -- MR. NIGH: Form objection. Form objection. Go ahead. You can answer. THE WITNESS: **So, you have -- you have pretty much one occupational study that I relied on, and that's in Hidajat.** And I talked about that extensively in more than a couple of pages. And then -- so that's obviously different than the epi studies."); *see* Ex. G., Hidajat *et al.*; *see generally* Report. Hidajat, however, suffers from serious limitations and should have been excluded based on Etminan's own

requirement that studies use "methodological or statistical means to control for confounding variables." Report at 13.

The Hidajat study is completely inapposite to the NDMA exposure at issue in this litigation. Hidajat studied nitrosamine exposure in a cohort of rubber factory workers in England between 1967 and 2015. As referenced above, Hidajat is an occupational study where the primary exposure to nitrosamines — along with numerous other carcinogens, including benzene — was through inhalation or skin contact over the course of decades, rather than oral ingestion over a few years. Tr. Vol. I, 201:6-10 (Q: So – but the Hidajat study as we discussed earlier today, was one of the occupational exposure where the exposure is primarily inhalation or contact through skin, right? A: Yes.").

Importantly, Hidajat did not control for smoking, a Class 1 known human carcinogen according to the International Agency for Research on Cancer (IARC). *See* Ex. G., Hidajat *et al.*, at 257; "List of Classifications, Agents classified by the IARC Monographs, Volumes 1–124," IARC Monographs on the Evaluation of Risk to Humans, *available at* https://monographs.iarc.who.int/list-of-classifications; Tr. Vol. I, 89:1-15 ("Q: Okay. And Hidajat, they did not directly control for smoking, right? A: They did not, although -- they did not, although they said they simulated smoking data, and the results did not change. Q: They simulated smoking data, but they didn't control for smoking, right? A: No. Q: Would you consider smoking to be

a potential confounder? A: So smoking is definitely a risk factor for cancer, and smoking, in order for it to be a confounder, has to also be potentially associated with NDMA exposure. So it could potentially be confounded, yes.").

Hidajat's failure to control for this key confounding variable substantially diminishes the study's value in examining any association between nitrosamines and cancer, much less any application to the NDMA exposure at issue here. Apparently recognizing this critical flaw, Dr. Etminan attempts to rehabilitate Hidajat by conducting an E-value statistical analysis to allegedly demonstrate that one confounder's presence is unlikely to change Hidajat's results. Report at 15. Among its many limitations, however, Hidajat, failed to control for other well-known cancer risk factors, such as family history of cancer and *H. pylori* infection. *See* Tr. Vol. I, 90:12-91:10. As Dr. Etminan admits, the E-value test he conducted does not work for multiple confounders. Tr. Vol. I, 225:12-225:21 ("Q: And just a minute ago, you were referring to this E-value methodology? A: Yes. Q: Magnitude of an unmeasured variable to reverse the risk, right? A: Yes. Q: What if there's two unmeasured variables? A: This methodology only works with one. It only works for one unmeasured confounder.") Thus, his E-value test is meaningless with regard to Hidajat.[2] Finally, as established during the deposition of one of Plaintiffs' other

[2] Dr. Etminan's misapplication of the E-value test calls into question the reliability of his entire analysis.

proffered general causation experts—Dr. Dipak Panigrahy—the cumulative levels of NDMA and total nitrosamine exposure examined in Hidajat are profoundly higher than those any Plaintiff could have consumed by orally ingesting valsartan. Ex. H, Panigrahy Tr. Vol. II, 486:7-488:21; 489:22-493:5; 499:10-17. Indeed, the annual nitrosamine exposures in Hidajat far exceed the cumulative nitrosamine exposures any Plaintiff could have reasonably received from taking affected valsartan. *Id.* Thus, Dr. Etminan's misplaced emphasis on Hidajat further renders his methodology unreliable. *See Zoloft*, 26 F. Supp. 3d at 485 (citing *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 158 (3d Cir. 1999)); *see also Pritchard*, 705 F. Supp. 2d at 485 ("[A] district court *may properly consider* the fact that an expert relied on few, if any, studies linking exposure to a particular illness in evaluating whether the expert had 'good grounds' to arrive at his conclusions.").

## III. DR. ETMINAN'S OPINIONS SHOULD BE EXCLUDED BECAUSE THEY DO NOT SHOW A RELIABLE CAUSAL RELATIONSHIP BASED ON THE EPIDEMIOLOGIC DATA.

The generally accepted method for determining whether there is a causal relationship between an exposure (NDMA and/or NDEA) and a disease outcome (cancers alleged) is to (1) look for statistically significant associations between NDMA and NDEA and cancer and, only when an association is found to exist, (2) then apply the Bradford Hill criteria. *See In re Johnson & Johnson Talcum Powder Prod. Mktg., Sales Pracs. & Prod. Litig.*, 509 F. Supp. 3d 116, 161 (D.N.J.

2020); *In re Roundup Prod. Liab. Litig.*, 390 F. Supp. 3d 1102, 1116 (N.D. Cal. 2018); *Zoloft*, 26 F. Supp. 3d at 455. If no association is found, mere chance has not been adequately ruled out as the explanation of the outcome (*i.e.*, cancer). At that point, the inquiry ends and the Bradford Hill criteria should not be applied to determine if there is a causal relationship. *McMunn v. Babcock & Wilcox Power Generation Grp., Inc.*, 2013 WL 3487560, at *15 (W.D. Pa. July 12, 2013) ("If no association between the exposure and the disease is supported by the scientific literature, there is no basis to find a causal relationship exists and the analysis should end there"); *Soldo*, 244 F. Supp. 2d at 461; *see also* Motion to Exclude Hecht at section I(A)(4).

When the analysis proceeds to application of the Bradford Hill criteria, "the 'techniques' used to implement the [Bradford Hill] analysis must be 1) reliable and 2) reliably applied.'" *Johnson & Johnson*, 509 F. Supp. 3d at 161 (quoting *Zoloft*, 858 F.3d at 796). On the whole, a Court must "assure itself that the expert's conclusions are not based upon unreasonable extrapolations from existing data." *Roundup*, 390 F. Supp. 3d at 1134 (citing *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). Dr. Etminan's opinions fail both parts of this analysis, as he did not reliably identify a statistically significant association and did not reliably apply the Bradford Hill criteria.

### A. Dr. Etminan's Methodology Ignores Widely Accepted Standards For Statistical Significance.

Instead of following well-established statistical standards to evaluate general causation, Dr. Etminan posits that statistical significance "doesn't have anything to do with causation." Tr. Vol. I, 29:12-20 ("Q: So statistical significance -- are you saying -- what I understand you to be saying is that statistical significance is not evidence of causation? . . . THE WITNESS: Yeah, it's -- statistical significance doesn't have anything to do with causation."). His rejection of this basic scientific and universally accepted standard alone renders his opinions unreliable. *See Zoloft*, 858 F.3d at 793 (concluding that statistical significance "remains an important metric to distinguish between results supporting a true association and those resulting from mere chance").

Moreover, Dr. Etminan's methodology disregards widely accepted standards that (1) a confidence interval inclusive of 1.0 denotes no association between exposure and outcome; and (2) a relative risk or odds ratio greater than 2.0 may be causal. *See* Reference Manual on Scientific Evidence, at 567, 582 (3d Ed. 2011); *see* Motion to Exclude Hecht at section I(A)(2). Instead, he creates his own statistical standard that a confidence interval inclusive of 1.5 or greater denotes *possible* causation:

> Q: Where are you coming up with this -- this limit of 1.5?
> A: 1.5 or higher, not just 1.5. 1.5 or higher. Because it's – it's technically a 15 percent increase that -- that is included in that interval.

> And one should, you know, do a further investigation to further look at that. I don't think it's – it's high enough to warrant further investigation with a bigger study, you know, higher number of cases. It is not a definitive negative with those numbers.

Tr. Vol. I, 159:9-20.

In other words, rather than recognize a statistical threshold that must be met to establish causation, Dr. Etminan hypothesizes that causation *may* exist where it cannot be disproven under his contrived standard. Thus, even where he admits the statistical data is "inconclusive," Dr. Etminan makes the illogical leap that a causal relationship exists between NDMA or NDEA and cancer because an increased risk "could not be ruled out." *Id.*; *see, e.g.*, Report at 20-21 ("As such the RR of 1.13, although not statistically significant, had an upper bound (of the confidence interval) of 1.68, which means a 68% increased risk of esophageal cancer could not be ruled out."). Dr. Etminan's distorted concepts of statistical significance and causation defy well-accepted scientific standards, and his opinions are not reliable for this reason.

**B. Dr. Etminan Applies His Own Criteria Inconsistently.**

Even if Dr. Etminan's stated methodology was reliable, he applies his own criteria inconsistently by relying upon studies where they support his preferred conclusions and criticizing those same studies where they do not. For example, Dr. Etminan criticizes the Knekt dietary study, which found no association between NDMA and gastric cancer, on the basis that it failed to account for comorbidities. Report at 17-18; *see* Ex. I, Knekt *et al*. In discussing NDMA and colorectal cancer,

however, Dr. Etminan relies on Knekt's finding of an association. Report at 19. Similarly, Dr. Etminan criticizes the Loh dietary study's findings for imprecise estimate of risk and small sample size with respect to gastric cancer, lack of control for prior history with respect to prostate cancer, and failure to control for competing risks with respect to lung cancer. Loh found no association with NDMA for each of these cancer types. *Id.* at 17, 23, 24; *see* Ex. J, Loh *et al*. But for colorectal cancer and head and neck cancers, Dr. Etminan relies on Loh's findings of an association without recognizing these same limitations. Report at 19-21. Finally, Dr. Etminan criticizes the Keszei dietary study's finding of no association between NDMA and gastric cancer based on "potential for misclassification (inaccurate reporting of different food intake by the subjects)." *Id.* at 17; *see* Ex. K, Keszei *et al*. This is a limitation of all dietary studies that Dr. Etminan fails to acknowledge in his report.[3] Yes, where Keszei (and the other dietary studies) find an association, statistically significant or otherwise, Dr. Etminan selectively overlooks this limitation and relies on the findings. *See, e.g.*, Report at 21.

Dr. Etminan's failure to employ a consistent methodological or analytical approach to the studies he relies on renders his methodology unreliable, and on this basis the Court should exclude his opinions.

[3] Dr. Etminan purports to rely upon 18 different dietary studies to support his opinions. Report at 15.

### C. Dr. Etminan Misapplies The Bradford Hill Criteria.

Dr. Etminan appears to fundamentally misunderstand both the overall purpose of a Bradford Hill analysis and many of the individual criterion. As a result, the conclusions he draws in his Bradford Hill analysis are not reliable.

Bradford Hill is a means by which epidemiologists analyze the totality of the evidence for causation ***after*** an association between exposure and outcome is found. *See Zoloft*, 858 F.3d at 795 ("The Bradford Hill criteria are metrics that epidemiologists use to distinguish a causal connection from a mere association."). Accordingly, at least one study showing an association between exposure and outcome must exist before a Bradford Hill analysis is conducted. Without establishing a statistically significant association for each of the nine cancers, however, Dr. Etminan improperly proceeds to the Bradford Hill causation inquiry. Report at 26-29. From there, he fails to distinguish between NDMA or NDEA or among the nine cancers and simply cherry-picks certain studies that support his ultimate conclusions that NDMA and NDEA cause all nine types of cancer. *Id.* This cursory analysis misapplies Bradford Hill.

For example, Dr. Etminan admits his conclusion that a dose-response relationship exists between NDMA and every type of cancer is "mainly driven by *Hidajat*." Report at 27-28. In doing so, he disregards Jakzyn (2006) and the numerous other studies that did not find a statistically significant association

between NDMA/NDEA and cancer in humans. Similarly, Dr. Etminan concludes that "[t]he strength of the evidence with respect to the risk of cancer . . . is considered high" based only on Hidajat and two other dietary studies, without any discussion of the studies that found no statistically significant association. *Id.* at 28. In analyzing the "analogy," criterion – whether similar compounds also cause the same outcome, Dr. Etminan cites only a single study finding an association between *nitrites* (a potential dietary precursor of NDMA) and cancer to conclude that "this criterion plays a significant role in the presence of a causal link between NDMA/NDEA in valsartan and cancer." *Id.* at 27. Most tellingly, Table 2 of Dr. Etminan's report, which he uses to demonstrate that seven of the nine Bradford Hill Criteria weigh in favor of causation for all nine cancers, is based on just one study – Hidajat. *Id.* at 29. As set forth above, in that study, the duration of exposure was far longer than any at issue in these cases and the mechanism of exposure was entirely different; NDMA exposure also was inseparable from the workers' exposures to known carcinogens, and the levels of exposure were exponentially higher.

Additionally, Dr. Etminan demonstrates a fundamental lack of understanding for some of the individual Bradford Hill criteria. In particular, he confuses "coherence" and "consistency" and treats them as interchangeable. *Compare* Report at 28 ("Consistency: This criterion asks whether there is a link or coherence between the basic scientific evidence and epidemiological evidence that draws a causal

link[.]"), *with id.* at 29 ("Coherence: Coherence examines whether there is a link or coherence between basic science and epidemiological evidence.").

As Dr. Etminan's conclusions drawn from his flawed Bradford Hill analysis are not reliably related to the totality of the available data, his causation opinions must be excluded.

## IV. DR. ETMINAN SHOULD BE PRECLUDED FROM OFFERING CERTAIN OPINIONS THAT ARE UNSUPPORTED BY ANY SCIENTIFIC EVIDENCE.

A number of Dr. Etminan's opinions with respect to certain cancers and exposure to either NDMA or NDEA are plainly unsupported by any scientific evidence and therefore unreliable under Rule 702. At a minimum, the Court should exercise its discretion to prune these unreliable opinions and preclude Dr. Etminan from offering them at any later hearing or trial. *See, e.g.*, *Johnson & Johnson*, 509 F. Supp. 3d at 176-77 (excluding portion of general causation experts' opinion because "the experts fail[ed] to give any scientific support" and "[the] Court need not 'admit any opinion evidence that is connected to existing data by only the *ipse dixit* of the expert'" (citation omitted)).

### A. Dr. Etminan Should Be Precluded From Offering Any Opinion That NDEA Causes Esophageal, Stomach, Colorectal, Liver, Lung, Bladder, Prostate, Blood, or Pancreatic Cancers.

Dr. Etminan's causation opinions with respect to NDEA are devoid of any support in the scientific literature. Specifically, Dr. Etminan does not demonstrate

that the evidence supports any association—let alone a causal association—between NDEA and the 9 cancers for which he purports to offer causation opinions. Because Dr. Etminan's conclusions with respect to NDEA and the cancers alleged find no basis in scientific evidence and the one study on which he relies does not bridge the gap, Dr. Etminan's opinions should be excluded.

Dr. Etminan could not cite—in his report or during his deposition—even one study demonstrating a statistically significant association between NDEA and esophageal, stomach, colorectal, liver, lung, bladder, prostate, or blood cancers. *See* Tr. Vol. II at 44:9-15 (breast cancer), 44:16-21 (esophageal cancer), 44:22-45:2 (stomach cancer), 45:3-8 (colorectal cancer), 45:9-14 (liver cancer), 45:15-20 (lung cancer), 45:21-25 (bladder cancer), 46:1-6 (prostate cancer), 46:7-12 (blood cancer). Because Dr. Etminan could not give any scientific support for his causal opinions between NDEA and these cancers, he should be precluded from offering any such opinions.

With respect to pancreatic cancer and NDEA, Dr. Etminan identified only one observational study published in the literature that found a statistically significant association between NDEA and pancreatic cancer: the Zheng study. Tr. Vol. II at 46:13-25; Report at 20; *see* Ex. L, Zheng *et al*. The authors of this study "found a biologically plausible positive association of . . . NDEA . . . and pancreatic cancer." *Supra*, Ex. L, Zheng *et al.* at 261.

However, the authors also expressly limited this finding by concluding that further research is needed to confirm a potential association: "In addition to a careful examination of potential mechanisms, these findings need to be confirmed in readily available large, prospective cohort studies with consideration of sufficient time between diet assessment to disease diagnosis or symptoms and diagnosis. ***If confirmed***, it will add direct evidence to the carcinogenic role of [NDEA] in human pancreatic cancer." *Supra*, Ex. L, Zheng *et al.*, at 261 (emphasis added); *id.* at 254 ("Although some of ***our findings probably reflect reverse causation bias*** due to lower meat intake in cases with latent disease, biologically plausible findings for pancreatic carcinogens, NDEA and NDMA, ***warrant further prospective investigation***." (emphasis added)).

Dr. Etminan expressly acknowledged during his deposition that (1) even while finding an association between pancreatic cancer and NDEA, the authors of the Zheng paper were careful to note that their observations were preliminary; and (2) Dr. Etminan, is not aware of any large, prospective cohort study like the authors of the Zheng paper recommended. Tr. Vol. II at 46:13-47:7, 47:20-48:15. Because the authors of the Zheng paper reported that further research needed to be conducted in order to confirm whether the results are indicative of a positive causation between NDEA and pancreatic cancer—and Dr. Etminan is not aware of any follow-up research having actually been conducted—Dr. Etminan cannot reasonably rely on

this study as the sole basis for a purported causal association between NDEA and pancreatic cancer. *See Johnson & Johnson*, 509 F. Supp. 3d at 164 ("[W]here the authors [of a study] found an association to not be statistically significant, an opinion may be unreliable.").

Dr. Etminan states in conclusory fashion, citing no authority, that based on NDEA's "carcinogenic profile" and "the substantial similarity in the mechanism of action of NDEA in causing cancer to NDMA," he would "expect[ ] that NDEA also has the potential to cause the other 8 cancers [in addition to pancreatic cancer] associated with NDMA." Report at 32. This opinion is an unreasonable extrapolation for which Dr. Etminan provides no scientific support. *See Roundup*, 390 F. Supp. 3d at 1134 (citing *Joiner*, 522 U.S. at 146) ("The Court must also assure itself that the expert's conclusions are not based upon unreasonable extrapolations from the existing data.").

The lack of studies linking NDEA to cancer in humans leaves "only 'the *ipse dixit* of the expert'" to support Dr. Etminan's conclusion." *Heller*, 167 F.3d at 155. This Court should consider that Dr. Etminan relies on no studies linking NDEA exposure to eight cancers and only one study, which the authors acknowledge was limited, linking NDEA exposure to pancreatic cancer. *Id.* at 158. Dr. Etminan's jump to compare NDEA to NDMA, based on nothing more than its "carcinogenic profile," is pure speculation unsupported by any authority. In the absence of any demonstrated

association between NDEA and these nine cancers, Dr. Etminan proceeded improperly when he further analyzed the (unproven) association and concluded it was causal under the Bradford Hill factors. *See McMunn*, 2013 WL 3487560, at *15 ("If no association between the exposure and the disease is supported by the scientific literature, there is no basis to find a causal relationship exists and the analysis should end there.").

Moreover, when confronted with a specific exemplar derived from the available data in this case, Dr. Etminan was unable to even offer an opinion, let alone cite to a single study, that supported a finding of causation. Tr. Vol. II, 49:5-20 ("Q: Can we agree that nowhere in your report you ever conclude that an increase in NDEA intake in the amounts contained in Mylan's valsartan-containing medications was sufficient to cause cancer in humans? A: Yes. Q: And you -- and in your work in this case, you have not found a single study in the peer-reviewed literature that would support a statistically significant increased risk of any cancer from a short-term duration nitrosamine intake increase of 150 nanograms per day, right? A: You mean a specific study that – that looks at that specific dosage and cancer? Q: Yes. A: No."). Thus, Dr. Etminan's generalized conclusion regarding a hypothetical association between NDEA and cancer is untethered from the facts of this case and should not be admissible to prove general causation. Dr. Etminan should be

precluded from offering baseless opinions that NDEA exposure causes any of the aforementioned cancers.

### B. Dr. Etminan Should Be Precluded From Offering Any Opinion That NDMA Causes Liver, Bladder, Prostate, Blood, Or Pancreatic Cancers.

Dr. Etminan's causation opinion with respect to NDMA and liver, bladder, prostate, and pancreatic cancer is not supported by scientific evidence. Specifically, Dr. Etminan relies only upon studies that show (a) no association or (b) no statistically significant association and the Hidajat study. Because Hidajat should have been discounted by Dr. Etminan according to his own criteria (*see supra* II.C), Dr. Etminan's causal opinions with respect to liver, bladder, prostate, and pancreatic cancers and NDMA exposure are left unsupported by any valid scientific evidence. As with his opinions related to NDEA, Dr. Etminan's opinions related to the causal relationship between NDMA exposure and liver, bladder, prostate, or pancreatic cancer is nothing more than the "the *ipse dixit* of the expert." *Heller*, 167 F.3d at 155. Because Dr. Etminan's opinions are not supported by scientific evidence, he should be precluded from offering these opinions.

With respect to liver cancer, Dr. Etminan relies only on the Straif study and Hidajat. *See* Report at 22; Ex. M, Straif *et al*. Straif, like Hidajat, examined the effects on rubber workers of occupational exposure to nitrosamines (as a class, not specifically NDMA) but failed to find any statistically significant association

between nitrosamines and liver cancer. *See* Ex. M, Straif *et al*. Dr. Etminan admits that Straif "only had 9 deaths due to liver cancer and lacked statistical power[,]" and that he did not find any "[d]ata from large epidemiological studies with respect to liver cancer that specifically quantified the levels of NDMA exposure and had an adequate follow up." Report at 22. Thus, Dr. Etminan cites no study at all supporting even a correlation between ingestion of NDMA and liver cancer. *See* Report at 22 (relying on Hidajat as the "strongest evidence" (and only evidence) of the risk of liver cancer). Notably, unlike the other types of cancers contained in his report, Dr. Etminan himself does not even include a concluding paragraph stating that it is probable that exposure to NDMA increases the risk of liver cancer. *Compare* Report at 22, *with* Report at 18-23. Accordingly, Dr. Etminan should be precluded from offering any opinion that NDMA increases the risk of liver cancer.

Next, with respect to bladder cancer, Dr. Etminan again relies only on two studies, Jakszyn *et al*. (2011) and Straif, which show a non-statistically significant association and no association, respectively, between NDMA and bladder cancer, to bolster the purported association through varied and inapplicable exposure routes allegedly demonstrated by Hidajat. *See* Report at 22, Ex. M, Straif *et al.*, Ex. N, Jakszyn *et al*. (2011). Dr. Etminan admits that the findings in Straif were not powerful enough to show any association but attempts to bolster these findings with Hidajat. *Id.* ("As with most findings in *Straif*, the data on bladder cancer was

also inconclusive[.] And yet again, as a result of *Hidajat* being a more well-powered and designated study . . . it was able to detect a statistically significant increase in the risk of bladder cancer deaths . . . while *Straif* was unable to produce statistically significant results."). The only other study Dr. Etminan relies upon in reaching his conclusion is Jakszyn, which did not find a statistically significant increased risk of bladder cancer with dietary NDMA exposure. *See* Report at 22 (citing Jakszyn (Ex. N)). Thus, Dr. Etminan should be precluded from offering any opinion that NDMA increases the risk of bladder cancer.

Dr. Etminan follows the same pattern with respect to prostate cancer. He relies on Loh, Straif and another Jakszyn study—all of which produced either no association or a statistically non-significant association. Report at 23; *see* Ex. J, Loh *et al*. (finding no association between the risk of prostate cancer and NDMA exposure); Ex. O, Jakszyn *et al*. (2012) (finding that NDMA was not statistically associated with the risk of prostate cancer); Ex. M, Straif *et al.* (finding no association between prostate cancer and NDMA exposure). Yet again, Dr. Etminan attempts to gloss over this evidence and rely only on Hidajat. Report at 22. Accordingly, Dr. Etminan should be precluded from offering any opinion that NDMA increases the risk of bladder cancer.

With respect to blood cancers, Dr. Etminan again provides scant scientific evidence upon which he bases his conclusion that NDMA exposure increases the

risk of blood cancers (lymphoma, leukemia, and multiple myeloma) in humans. Dr. Etminan relies on the Richardson study, which produced a statistically significant result but between "nitrites, nitrates, [and] nitrosamines (all three combined) as one entity" and lymphoma. Report at 23; *see also* Ex. P, Richardson *et al*. Otherwise, Dr. Etminan relies only on the Straif study, which found no association between NDMA and blood cancers. Report at 23. That again leaves Dr. Etminan relying on Hidajat's conclusion with respect to lymphoma — which is not based on the totality of the evidence and has nothing to do with any other blood cancer — to support his conclusion as to "blood cancers" and NDMA. Dr. Etminan should be precluded from offering any opinion that NDMA increases the risk of any blood cancers, but particularly leukemia and multiple myeloma for which Dr. Etminan cannot cite a single study in support.

Finally, Dr. Etminan's opinion that NDMA exposure increases the risk of pancreatic cancer is unreliable. *See* Report at 19-20. Dr. Etminan relies only on two studies in addition to Hidajat in an attempt to reach his conclusion. Specifically, Dr. Etminan relies on the Fritschi study (Ex. Q, Fritschi *et al*.), which he acknowledges found no association between nitrosamines (again, not NDMA specifically) and pancreatic cancer (Report at 19), and Straif, which found no association between pancreatic cancer and NDMA exposure (Ex. M, Straif *et al*.). Dr. Etminan attempts to rely on one portion of the Zheng study, which for the reasons set forth above also

does not support Dr. Etminan's conclusion, *see supra* at 25-27, but Zheng did not find a statistically significant association between NDMA and pancreatic cancer. *See* Ex. L, Zheng *et al.*; *see* Report at 20 (acknowledging NDMA exposure (from plant and animal sources) did not reach statistical significance with respect to pancreatic cancer). Thus, once again, Dr. Etminan cites only Hidajat as support for his conclusion that NDMA exposure causes an increased risk of pancreatic cancer. Report at 19-20. As such, Dr. Etminan should be precluded from offering any opinion that NDMA increases the risk of pancreatic cancer.

In sum, Dr. Etminan should be precluded from offering any opinion that NDMA exposure increases the risk of liver, bladder, prostate, blood, or pancreatic cancers.

## CONCLUSION

For all of the foregoing reasons, Defendants respectfully request that the Court exclude Dr. Etminan's opinions.

Date: November 1, 2021

Respectfully Submitted:

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**CERTIFICATE OF SERVICE**

I HEREBY CERTIFY that on November 1, 2021, I electronically filed the foregoing with the Clerk of the Court by using the CM/ECF system which will send a notice of electronic filing to all CM/ECF participants in this matter.

*/s/ Seth A. Goldberg*
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